



UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER OF PATENTS AND TRADEMARKS
Washington, D.C. 20231
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/550,605	04/17/2000	Leif Andersson	064727.0109	1231

26118 7590 12/04/2002

BROBECK, PHLEGER & HARRISON, LLP
ATTN: INTELLECTUAL PROPERTY DEPARTMENT
1333 H STREET, N.W. SUITE 800
WASHINGTON, DC 20005

EXAMINER

SOUAYA, JEHANNE E

ART UNIT	PAPER NUMBER
----------	--------------

1634

DATE MAILED: 12/04/2002

19

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

09/550,605

Applicant(s)

Andersson et al

Examiner

Jehanne Souaya

Art Unit

1634



-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136 (a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on Sep 6, 2002.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1 and 3-17 is/are pending in the application.
- 4a) Of the above, claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1 and 3-17 is/are rejected.
- 7) ☒ Claim(s) 10 is/are objected to.
- 8) ☐ Claims _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved by the Examiner.
If approved, corrected drawings are required in reply to this Office action.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

- 13) ☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
a) ☐ All b) ☐ Some* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
2. ☐ Certified copies of the priority documents have been received in Application No. _____.
3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
*See the attached detailed Office action for a list of the certified copies not received.
- 14) ☒ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).
a) ☐ The translation of the foreign language provisional application has been received.
- 15) ☒ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892) 4) ☐ Interview Summary (PTO-413) Paper No(s). _____
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948) 5) ☐ Notice of Informal Patent Application (PTO-152)
- 3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s). _____ 6) ☐ Other: _____

Art Unit: 1634

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed September 6, 2002 (request for RCE) and the amendment after final, submitted July 8, 2002, has been entered.
2. Currently, claims 1 and 3-17 are pending in the instant application. All the amendments and arguments have been thoroughly reviewed but are deemed insufficient to place this application in condition for allowance. Any rejections not reiterated are hereby withdrawn. The following rejections are either newly applied or are reiterated. They constitute the complete set being presently applied to the instant Application. Response to Applicant's arguments follow.
3. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

Art Unit: 1634

Maintained Rejections

Written Description

4. Claims 14 is rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The claim is drawn to determining whether a pig has a white coat color by detecting a sample of pig KIT protein to determine whether the protein is a splice variant protein, wherein said protein is correlated with white coat color. The specification teaches that an alteration of the 5' intron splice site of intron 17 of the KIT2 gene is associated with the white coat color of pigs. The specification teaches that the alteration is from a GT pair to an AT pair which affects the splicing of the pre mRNA and results in the loss of the whole of exon 17 from the mRNA transcribed from the I-KIT2 sequence. It is further noted that the porcine KIT protein has at least 19 exons, whereas the specification has only taught a single splice variant (a protein lacking exon 17) and has only correlated white coat color with said single splice variant. The specification fails to describe a representative number of the various splice variants encompassed by the claims, such as proteins lacking a single or multiple exons, and further fails to demonstrate an association between these numerous undisclosed splice variants and white coat color in a pig. The disclosed structural splice variant protein lacking exon 17 does not constitute a substantial portion of the genus of proteins for use in the method claimed. As set forth by the Court in *Vas*

Art Unit: 1634

Cath Inc. V. Mahurkar, 19 USPQ2d 1111, the written description much convey to one of skill in the art "with reasonable clarity" that as of the filing date applicant was in possession of the claimed invention. Absent a written description disclosing a representative number of the species of KIT splice variant proteins associated with white coat color in a pig, the specification fails to show that applicant was in fact, "in possession of the claimed invention" at the time the application for patent was filed.

Response to Arguments

The response does not address the rejection made with respect to claim 14.

New Grounds of Rejection and Objection

Claim Objections

5. Claim 10 is objected to under 37 CFR 1.75© as being in improper form because a multiple dependent claim cannot depend from a claim which is dependent on another multiply dependent claim (claim 8 is dependent on claim 5 which is dependent on a multiply dependent claim). See MPEP § 608.01(n).

Indefinite

6. Claims 1, 3-17 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Art Unit: 1634

Claims 1 and 15 are indefinite in the recitation of “presence or absence of said mutation is correlated with white coat color” because it is unclear how the presence and absence of a mutation can be correlated with white color, when the specification teaches that the presence of a G to A substitution in the first nucleotide of intron 17 of the 2nd copy of the porcine KIT gene is responsible for the white coat color phenotype in pigs. Furthermore, the last positive process step of the claim teaches analyzing a nucleic acid to determine whether a mutation is present at exon 17/intron 17 splice site of KIT gene, which does not relate back to the preamble which states “a method for determining whether a pig has a white coat color”, such that it is unclear if the method is drawn to detecting whether a pig has a white coat color, or detecting a mutation at exon 17/intron 17 splice site of KIT gene. It is noted that the claim does not recite how to determine whether a pig has a white coat color, as stipulated in the preamble of the claim.

Claim 14 lacks sufficient antecedent basis for the recitation of “the splice variant protein” as the term “splice variant protein” was not recited previously in the claim. It is noted that a preliminary amendment filed 1/29/2001 changed “the splice...” to --a splice...--, however this amendment is not present in the amended claims of the July 08, 2002, response.

Claim 14 lacks sufficient antecedent basis for the recitation of “said protein” because it is unclear whether this recitation refers to the “pig KIT protein”, or the “splice variant protein” previously recited in the claim.

Art Unit: 1634

Enablement

7. Claims 1 and 3-17 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for 1) a method or kit for determining whether a pig has a white coat color which comprises obtaining a sample of pig nucleic acid and analyzing the nucleic acid obtained to determine whether a substitution of the G of the conserved GT pair with A at the exon17/intron17 splice site of KIT2 wherein presence of said substitution is correlated with coat color or 2) a method or kit for determining whether a pig has a white coat color which comprises obtaining a sample of pig nucleic acid and analyzing the nucleic acid obtained to determine whether a mutation exists at the exon 17/intron 17 splice site of KIT2 that results in a pig KIT mRNA lacking exon 17, wherein the presence of said mutation is correlated with white coat color in a pig, or 3) to a method for determining whether a pig has a white coat color by analyzing a sample of pig KIT protein to determine whether a splice variant KIT protein missing exon 17 is present, wherein a ratio of normal KIT protein and said splice variant KIT protein missing exon 17 of 1:1, 2:1, or 3:1 is correlated with white coat color, does not reasonably provide enablement for a method for determining coat color genotype in a pig by determining whether 1) any mutation is present at exon17/intron17 boundary of KIT gene, wherein presence or absence of the mutation is correlated with coat color or 2) any splice variant protein present being correlated with coat color. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims.

Art Unit: 1634

The claims are broadly drawn to methods and kits for determining coat color genotype in a pig by determining whether a mutation is or is not present at an exon17/intron 17 splice site, wherein presence or absence of the mutation is correlated with coat color and to methods for determining whether a pig has a white coat color by correlating any splice variant KIT protein with white coat color. Firstly, it is noted that the claims appear to indicate that both the presence and absence of the mutation is correlated with white coat color. However, the specification teaches that the presence of an alteration at the conserved GT pair (G to A mutation) affects the splicing of the pre mRNA and results in the loss of the whole of exon 17 from the mRNA transcribed from the I-KIT2 sequence. The specification further teaches that the absence of splice KIT mutants are correlated with patched or colored phenotypes. Secondly, the specification does not teach correlating coat color to any of the various substitutions, insertions, deletions or frame shift mutations to the exon 17/intron 17 splice site that are encompassed by the claims. Neither the specification nor the claim make clear that "exon17/intron 17 splice site" only refers to the conserved GT pair at the beginning of intron 17. Conserved sequences important for proper splicing of introns are present at both the 5' end and the 3' (acceptor) end of an intron as well as sequences within the intron. However, the specification does not teach mutations in either this region or the 3' region of intron 17 that would lead to a deletion in exon 17. The specification teaches that consequences for mutation occurring within a splice site may be a reduction in the amount of mRNA produced and/or utilization of alternative but incorrect splice sites in the vicinity, resulting in a production of mRNA which either contains additional

Art Unit: 1634

intron sequences or which may lack a portion of coding sequence (p. 4, lines 20-26). The specification differentiates such mutations from mutations at the 5' (donor) splice which prevent binding of protein factors and lead to the exon not being recognized and therefore excised along with neighboring introns. The specification only demonstrates the latter type of mutation, which is the mutation of a G to an A at the conserved GT pair of the 5' donor splice site of intron 17 which results in white coat color in a pig. The specification, however, provides no guidance as to the effect the myriad of other mutations that could occur in the exon 17/intron 17 splice site, would have on the KIT mRNA or protein, nor how this would affect the coat color of a pig. Additionally, the specification does not teach what the coat color of a pig would be if only a single copy of the KIT gene is present and possesses the G to A substitution in the conserved GT pair.

The art does not teach of a correlation between mutations in the KIT gene and coat color of a pig. Since it is unclear from the teachings in the specification or the art as to how mutations in the KIT gene affect coat color in a pig, it is unpredictable as to whether the myriad of mutations in either one or a second or both copies of the KIT gene encompassed by the broadly claimed invention would affect coat color in a pig or how they would affect coat color in a pig. Neither the specification nor the art teach a predictable correlation between the large number of possible mutations in a single KIT gene, or the 2nd of two KIT genes, or 2 KIT genes encompassed by the claims and coat color in a pig. The specification only teaches of a single substitution from a G to an A in the conserved GT pair at the exon17/intron17 splice site in the

Art Unit: 1634

second copy of KIT gene that results in a single splice variant KIT protein lacking exon 17 and results in a pig with white coat color. With regard to claim 14, the porcine KIT gene contains at least 19 exons. Splice variant proteins encompassed by the claim include variants lacking single or multiple exons or parts of exons of the KIT protein, however, the specification has not provided any guidance as to the effect that the large number of splice variants encompassed by the claim, would have on the coat color of a pig. The single mutation which leads to the single splice variant protein lacking exon 17 taught by the specification is not sufficient to establish a predictable correlation for the skilled artisan between the large number of mutations that might result in an altered exon 17 (not necessarily completely excised, as discussed by the specification at page 4) or splice variant proteins encompassed by the claims and white coat color of a pig. Further, the specification provides no guidance as to how the splice variant protein lacking exon 17 leads to white coat color in a pig, such that the skilled artisan might be able to establish a predictable correlation between the effect the lack of exon 17 has on the function of the porcine KIT protein and other splice variants of the porcine KIT protein. As correlating a particular coat color with the large number of mutations in the KIT gene encompassed by the broadly claimed invention is unpredictable in light of the lack of teaching and guidance in the specification and the art, the skilled artisan would be required to perform undue experimentation to make or use the invention as broadly as it is claimed. While the amount of experimentation needed is not necessarily considered undue, such experimentation would be replete with trial and error, thus constituting undue experimentation.

Art Unit: 1634

Response to Arguments

The response traverses that the office action unnecessarily limits the claim scope to one mutation and that the specification sets forth how to determine whether a mutation has occurred. This argument has been thoroughly reviewed but was found unpersuasive. While the skilled artisan would be able to determine whether a mutation has occurred, as discussed above, the claims encompass determining whether a pig has a white coat color depending on the presence or absence of a mutation in the exon17/intron 17 splice site of KIT gene. While the specification teaches one mutation, which leads to a single specific splice variant protein, and when present, is correlated with white coat color, the specification does not provide any guidance as to other splice variant proteins that would also be correlated with white coat color, or how mutations in the exon 17/intron 17 splice site, that did not lead to the excision of exon 17, would effect the coat color of a pig. Case law has established that “(t)o be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without ‘undue experimentation.’” *In re Wright* 000 F.2d 1557, 1561. *In re Fisher*, F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970) it was determined that “(t)he scope of the claims must bear a reasonable correlation to the scope of enablement provided by the specification to persons of ordinary skill in the art”. The amount of guidance needed to enable the invention is related to the amount of knowledge in the art as well as the unpredictability in the art. Furthermore, the Court in *Genetech Inc. V Novo Nordisk* 42 USPQ2d 1001 held that “(I)t is the specification, not the knowledge of one skilled in the art that must supply the novel aspects of the invention in order to

Art Unit: 1634

constitute adequate enablement". With regard to the instant invention, the single mutation which leads to the single splice variant protein lacking exon 17 taught by the specification is not sufficient to establish a predictable correlation for the skilled artisan between the large number of mutations that might result in an altered exon 17 (not necessarily completely excised, as discussed by the specification at page 4) or splice variant proteins encompassed by the claims and white coat color of a pig. Further, the specification provides no guidance as to how the splice variant protein lacking exon 17 leads to white coat color in a pig, such that the skilled artisan might be able to establish a predictable correlation between the effect the lack of exon 17 or altered exon 17 has on the function of the porcine KIT protein and other splice variants of the porcine KIT protein. Given the lack of guidance in the specification and the art as outlined above, the trial and error analysis that the skilled artisan would have to perform to practice the invention as broadly as it is claimed is unpredictable, thus constituting undue experimentation. The examiner does not agree that the necessary experimentation is merely routine, because neither the art nor the specification teach how a single copy of pig KIT mRNA lacking exon 17 or other splice variant KIT proteins or KIT proteins which lack only parts of exon 17 or include additional intron sequences (as discussed by the specification at page 4), would affect the coat color of a pig. The specification provides no basis for predictable correlation between the effect the single disclosed mutation has on the coat color of a pig and the effect the myriad of possible mutations or splice variant proteins that are encompassed by the claims would have on the coat color of a pig.

Art Unit: 1634

Claim Rejections - 35 USC § 102

8. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless -

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

9. Claim 15 is rejected under 35 U.S.C. 102(b) as being anticipated by New England Biolabs (1996 catalog, p. 42).

Claims 15 is drawn to a kit comprising one or more reagents suitable for determining whether a mutation is present at an exon17/intron 17 splice site of the KIT gene. The claim encompasses a kit containing a single restriction enzyme.

New England Biolabs teaches the *Nla*III restriction enzyme in kit format (commercially available product packaged in container containing a buffer). The *Nla*III enzyme cleaves nucleotide sequences at CATG sites. When the G of the first nucleotide of intron 17 of the porcine KIT gene is mutated to an A, a CATG site occurs with the last nucleotide of exon 17 and the first 3 nucleotides of intron 17 in the genomic DNA of porcine KIT, therefore, the ability to recognize that mutation is an inherent property of the *Nla*III restriction enzyme. It is noted that the intended use for the kit outlined in claim ¹⁵~~17~~ carries no patentable weight.

98

Art Unit: 1634

Claim Rejections - 35 USC § 103

10. Claims 15 and 16 are rejected under 35 U.S.C. 103(a) as being unpatentable over Moller et al., (Mammalian Genome, 1996, vol. 7, pp 822-830) in view of Ohler et al (PCR Methods and Applications, vol. 3, 1993; pp 115-119) and further in view of Ahern ("Biochemical Reagent Kits Offer Scientists Good Return on Investment", pp 1-5, from The Scientist, vol: 9, page 20, 1995).

Claims 15 and 16 are drawn to a kit comprising one or more reagents suitable for determining whether a mutation is present at an exon17/intron 17 splice site of the KIT gene and reagents for carrying out a PCR reaction. Such a kit reads on nucleic acid primers that will amplify any fragment of the KIT gene, as long as the exon 17/intron 17 splice site is included. Moller (1996) teaches primer (see table 1) pairs that would amplify different portions of the KIT gene.

Ohler teaches that Long range PCR offers an alternative to isolation of genomic or cDNA clones from tissues, and that the ability to specifically amplify and detect PCR products ranging in size from 1 to 10 kb would facilitate several tasks in human genome research and that generation of a wide range of PCR products would potentially expedite isolation of uncloned DNA (gaps) represented in physical maps and provide a means for maintaining order and orientation of closely linked loci during analysis.

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to use the primers taught by Moller in a long range PCR taught by Ohler for the purpose of generating PCR products that could expedite isolation of uncloned DNA

Art Unit: 1634

represented in physical maps. The skilled artisan would have instantly recognized, for example, that primers KIT3F and KIT7R would be suitable for generating several kb of sequence from the KIT gene in long range PCR, including a portion of exon 2 through a portion of exon 19, including intervening introns (this includes the exon 17/intron 17 splice site), and that such would be useful in generating products that could expedite isolation of uncloned DNA represented in physical maps since, it is noted that at the full length porcine KIT gene sequence was not known.

Although neither Moller nor Ohler teach primers or reagents for PCR in kit format, it would have been obvious to package these primers (primers in table 1- the claims are not limited to a single primer pair) in kit format for a PCR reaction, including reagents for PCR, to make the invention of Moller in view of Ohler easier to perform because Ahern teaches that kits accelerate the research process, are convenient and save time (p. 4). As the use for a kit carries no patentable weight, the primers taught by Moller read on claim 15 and 16 regardless of why they were designed.

Response to Arguments

The response will be addressed as it applies to the new grounds of rejection. The response traverses that to arrive at the recited claim, the examiner chose two primers from the 14 primers listed in Table 1, with no other guidance than the present claims. This argument has been thoroughly reviewed but was found unpersuasive. Firstly, In response to applicant's argument that the examiner's conclusion of obviousness is based upon improper hindsight reasoning, it

Art Unit: 1634

must be recognized that any judgment on obviousness is in a sense necessarily a reconstruction based upon hindsight reasoning. But so long as it takes into account only knowledge which was within the level of ordinary skill at the time the claimed invention was made, and does not include knowledge gleaned only from the applicant's disclosure, such a reconstruction is proper. See *In re McLaughlin*, 443 F.2d 1392, 170 USPQ 209 (CCPA 1971). Secondly, the instant rejection sets forth the teaching of Ohler as motivation to use the primers from table 14. The use for the primers as stated in the rejection above, is different than the intended use set forth in the claims. The limitations in the claims are insufficient to overcome the teachings of Moller in view of Ohler and further in view of Ahern. The claims only recite reagents "suitable" for determining whether a mutation is/is not present at an exon 17/intron 17 splice site and undisclosed primer pairs (furthermore, the claims are not limited to a single primer pair). The teachings of Moller in view of Ohler and further in view of Ahern teach such limitations.

11. Claim 16 is rejected under 35 U.S.C. 103(a) as being unpatentable over Kuiper et al (US Patent 6,218,119, 102(e) date: 1/16/1996).

Claims 16 is drawn to a kit comprising one or more reagents suitable for determining whether a mutation is present at an exon17/intron 17 splice site of the KIT gene, a reagent for carrying out PCR, and a pair of suitable primers. The claim encompasses a kit containing a restriction enzyme that recognizes a CATG site (when the G of the first nucleotide of intron 17 of the porcine KIT gene is mutated to an A, a CATG site occurs with the last nucleotide of exon 17

Art Unit: 1634

and the first 3 nucleotides of intron 17 in the genomic DNA of porcine KIT), a polymerase, and a pair of primers that can amplify the CATG site. The intended use for the kit recited in claim 16 carries no patentable weight. Further, the kit does not require that the primers amplify porcine KIT sequences.

Kuiper et al teach a method for amplification of simple sequence repeats (see abstract, and example 5, cols 13-15). Kuiper et al specifically teach such a method using the enzyme *Nla*III which recognizes a CATG site and a pair of primers (see col 15, *Nla*III primer 1a and *Nla*III primer 1b) and the enzyme Taq-polymerase (col. 10, line 18) for use in the method. Kuiper et al further teaches a kit comprising a pair of primers for use in a method of amplifying sequence repeats (see claims 1-6). Although Kuiper et al do not specifically teach primers *Nla*III primer 1a and *Nla*III primer 1b, the *Nla*III restriction enzyme, or Taq polymerase in kit format, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to include such reagents in the general kit taught by Kuiper et al, to provide preweighed, premeasured reagents in kit format to minimize sample handling. The ordinary artisan would have been motivated to include such reagents in kit format because it would have been immediately obvious to the ordinary artisan that doing so would make the method of Kuiper et al (example 5) more convenient to perform.

Conclusion

12. No claims are allowable.

Art Unit: 1634

13. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Souaya whose telephone number is (703)308-6565. The examiner can normally be reached Monday-Friday from 9:00 AM to 6:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Jones, can be reached on (703) 308-1152. The fax phone number for this Group is (703) 305-3014.

Any inquiry of a general nature should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Jehanne Souaya

Jehanne Souaya
Patent examiner
Art Unit 1634

12/2/02